Motor Unit Action Potentials as a Source of Noise in the Non-Invasive Detection of Fibrillation Potentials

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Abstract— **Denervated muscle fibers produce spontaneous depolarizations termed fibrillation potentials. These potentials are an indicator of neuromuscular pathology and are detected by inserting a needle electrode into the muscle of interest to detect the time-based signal. A proposed non-invasive method measures the spectral energy corresponding to increased spontaneous muscle activity [1]. This paper exams the impact of normal muscle activity on such a method through the use of a computer model of fibrillation potentials and normal motor unit action potentials. A mathematical expression for the surface recorded signal is proposed and used as the basis for analyzing the temporal and spectral characteristics of spontaneous and normal motor activity. Based on these results, filtering methods for the removal of normal motor activity are proposed and future work needed to implement non-invasive detection of fibrillation potentials is discussed.**

Keywords— **Fibrillation potentials, electromyography, motor unit action potential.**

I. Introduction

FIBRILLATION potentials consist of the asynchronous firing of denervated individual muscle fibers [2]. Muscle fibers whose controlling motoneuron has been transected, crushed, or damaged in a variety of other pathologic processes, undergo physiologic changes in which the fiber membranes become unstable and begin to depolarize spontaneously [3]. The presence of fibrillation potentials is a useful diagnostic for the determination of neuromuscular pathology [2]. Needle electromyography (NEMG), which consists of inserting a needle electrode into the muscle of interest, is the standard method for detecting spontaneous muscle activity.

The invasive nature of NEMG, in addition to its associated costs and requirement for specially trained health care professionals, limits its use as a diagnostic. A recently proposed non-invasive method utilizing surface electrodes has the potential to solve some of these difficulties and may result in new applications, such as monitoring patient response to treatment [1]. Whereas NEMG detects the time domain spontaneous activity of individual muscle fibers, surface electrodes sample the summed activity of thousands of fibers. The resultant surface signal lacks discernible time domain features due to the small amplitude of individual fiber extracellular potentials. Fibrillation potentials are detected by measuring the spectral energy in frequency bands corresponding to muscle activity and comparing the energy between denervated and normal muscles.

While this method has been demonstrated on an animal model of denervation, additional challenges remain before this method can be applied to humans. Specifically, human patients often present with a complicated clinical picture of denervation with normal motor activity present in a muscle of interest in addition to underlying spontaneous muscle activity. The presence of healthy motor activity has the potential to complicate the detection of fibrillation potentials via spectral methods due to the additional spectral energy present in physiological frequency bands. This paper examines the impact of normal motor activity, in the form of motor unit action potentials (MUAPs), on the non-invasive detection of fibrillation potentials.

II. Modeling

A computer model of the human thenar muscle group was developed in order to examine the temporal and spectral characteristics of MUAPs and fibrillation potentials. The development of the model, the geometric distribution of muscle fibers, and the selection of model parameters have been previously described [1]. The basis of the model is the calculation of the extracellular potential generated in the firing of a single muscle fiber. This potential is calculated using the dipole model proposed by Dimintrova [4] and is given by:

$$\phi(p) = \frac{V_m \sigma_a d^2}{4\sigma_o} \sum_{i=1}^{N} \left\{ -\frac{x+vt-S_i}{[(x+vt)^2+y^2+z^2]^{3/2}} + \frac{x+vt-b+M_i}{[(x+vt-b)^2+y^2+z^2]^{3/2}} - \frac{x-vt+b-M_i}{[(x-vt+b)^2+y^2+z^2]^{3/2}} + \frac{x-vt+S_i}{[(x-vt)^2+y^2+z^2]^{3/2}} \right\} \quad (1)$$

where V_m is the change in membrane potential during an action potential, σ_a is the conductivity of the axoplasm, σ_o is the conductivity of the extracellular fluid, d is the diameter of the fiber, b is the length of the depolarization zone, and v is the conduction velocity of the depolarization. The fiber is assumed to be oriented longitudinally along the x-axis and the position of the fiber in relation to the recording electrode is given by the x, y, and z coordinates.

In order to generate a smooth action potential, N=200 linearly spaced dipoles were used to model the moving depolarization zone of each fiber. The shaping vector B was

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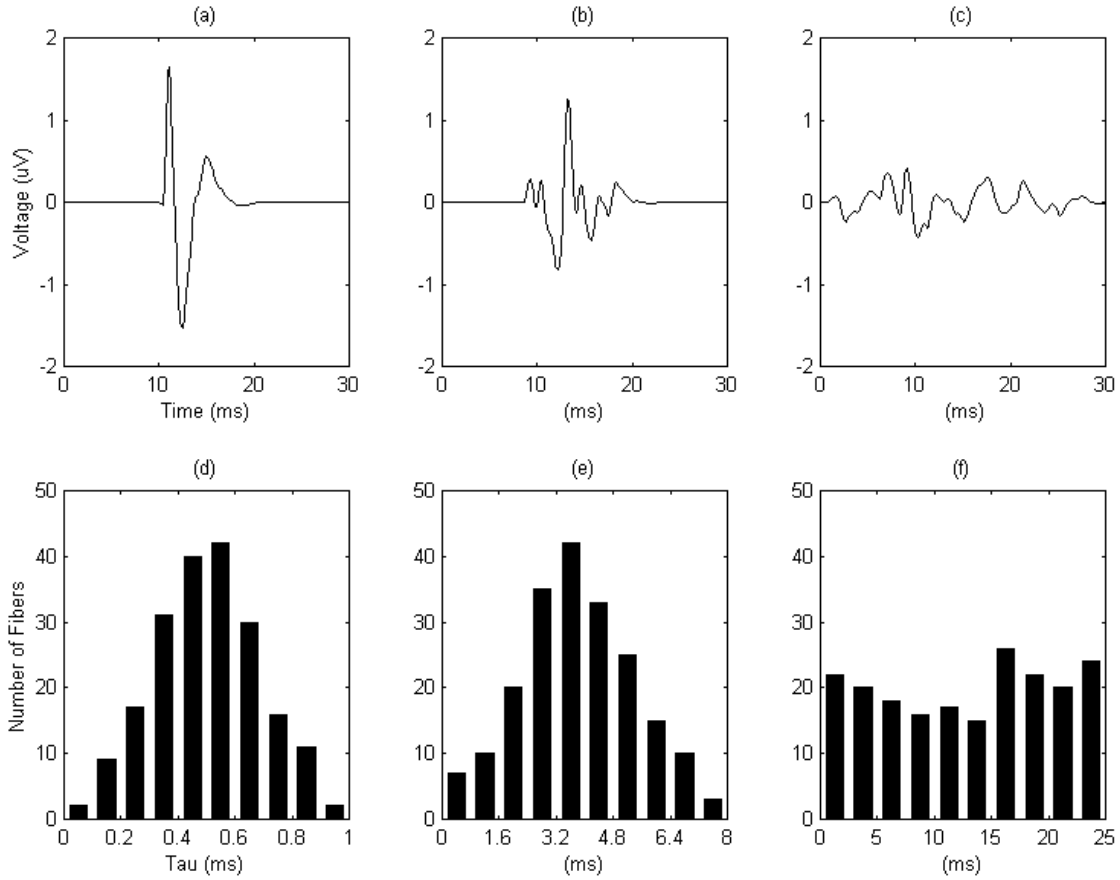

Fig. 1. Effect of varying distributions of the temporal dispersion, τ, on the morphology of the summed extracellular potential from 200 muscle fibers. (a)-(c) shows the extracellular potentials for the distributions shown in the histograms in (d)-(f), respectively. (a) models the dispersion of a normal motor unit action potential. (b) models the result of increased dispersion as seen in demyelinating polyneuropathies. (c) models the uniform firing distribution corresponding to fibrillation potentials.

linearly distributed over the first 50 percent of the depolarization zone and the trailing dipole shaping vector, M, was distributed over the final 45 percent. These shaping vectors model the gradual depolarization that occurs in real muscle fibers and provides for better accurracy than using only single leading and trailing dipoles to represent the boundaries of the depolarization zone. The shaping vector distributions were based on empirical estimation so that the morphology of modeled fiber action potentials resembled that of experimentally observed fiber action potentials. As each leading dipole reached the terminal end of a fiber, its corresponding trailing dipole was negated to result in a smooth reduction of the action potential as it dissipated.

A population of 30,000 fibers was generated to represent the thenar muscles from which a group of 200 randomly selected fibers was chosen to represent a single motor unit. The number of fibers in a motor unit was approximated from studies of the innervation ratio in human skeletal muscle [5]. For calculation of the power spectrum, sequences of motor activity and fibrillation potentials were generated. The potentials for a group of 5000 randomly selected fibers, organized into motor units for the model of normal motor activity, were calculated a duration of 1 second. The extracellular potential was calculated at two different arrays of twelve points each to model recording from bipolar surface electrodes.

III. Signal Analysis

Surface recordings of bioelectrical activity in skeletal muscle with bipolar electrodes can be considered to consist of varying levels of four different components: (1) fibrillation potentials; (2) normal motor activity; (3) powerline noise; and, (4) ambient and electrical noise. The first component, fibrillation potentials, is the signal of the interest, and the remaining three components are various types of noise. The powerline and ambient noise can be limited by the use of band and notch filters, low noise electrical components, input amplifiers with a high common mode rejection ratio, and minimizing the skin-electrode impedance [6], [7], [8]. Before developing filtering techniques to remove normal motor activity, it is important to first understand the characteristics of the extracellular potentials generated by MUAPs and fibrillation potentials.

A MUAP is initiated by its motoneuron resulting in the

synchronized firing of the individual fibers of that motor unit. Once that motor unit is denervated, the individual muscle fibers begin to fire spontaneously without synchronization. Previous studies have shown that fibrillation potentials are initiated in the motor end plate region of the fiber [9]. This results in the same extracellular potential being detected for a fiber regardless of whether the action potential was spontaneous or initiated by a motoneuron. If the extracellular potential for a given fiber is described as a basis vector, $b(t-\tau)$, then the potential generated by a group of fibers will differ in their temporal dispersion, τ, if they are firing synchronously as a motor unit or independently as fibrillation potentials. The resultant surface recorded signal can be modeled as:

$$s(t) = \sum_{i}^{N} \alpha_i b(t-\tau_{1i}) + \sum_{j}^{M} \beta_j b(t-\tau_{2j}) + p(t) + w(t) \quad (2)$$

where α_i and β_j are scaling factors that account for the size of a fiber and its distance from the recording electrodes, N is the number of denervated fibers, M is the number of fibers in the motor unit, τ_1 and τ_2 represent the time placement of each fiber potential for the denervated and normal fibers, respectively, p(t) is the powerline noise, and w(t) is the ambient noise. The motor activity term can be expanded to $\sum_k^P \sum_j^M \beta_{kj} b(t-\tau_{kj})$ to account for the firing of multiple motor units. P represents the number of motor units with specific values and distributions of β and τ, respectively, for each motor unit.

IV. Results

The distribution of the individual τ values for the denervated and normal fibers determines the temporal dispersion of the summation of the potential for each fiber population. Fig. 1 illustrates the effect of different distributions of τ values on the morphology of a motor unit action potential for a population of 200 fibers. The narrow Gaussian distribution of Fig. 1d, with a range of only 1 ms, results in the triphasic waveform of Fig. 1a which corresponds to a normal motor unit. The disorganized waveform of Fig. 1b results from the wider Gaussian distribution of Fig. 1e, representing a temporal dispersion of 8 ms. The increased temporal dispersion corresponds to demyelinating polyneuropathies that result in greater variation in the activation of individual fibers in a motor unit. The uniform distribution of Fig. 1f, modeling the temporal distribution of fibrillation potentials, produces a waveform lacking discernible features in comparison to a normal motor unit.

Fig. 2 shows the result of adding a Gaussian white noise sequence with a standard deviation of 0.3 μV to the fibrillation potentials and to a sum of the MUAP and fibrillation potentials. While the noise obscures the presence of the fibrillation potentials, the MUAP is clearly discernible.

Fig. 3 shows the power spectrum calculated from computer model generated sequences of normal MUAPs and fibrillation potentials. The MUAPs were randomly placed throughout the 1 second sequence while the fibrillation

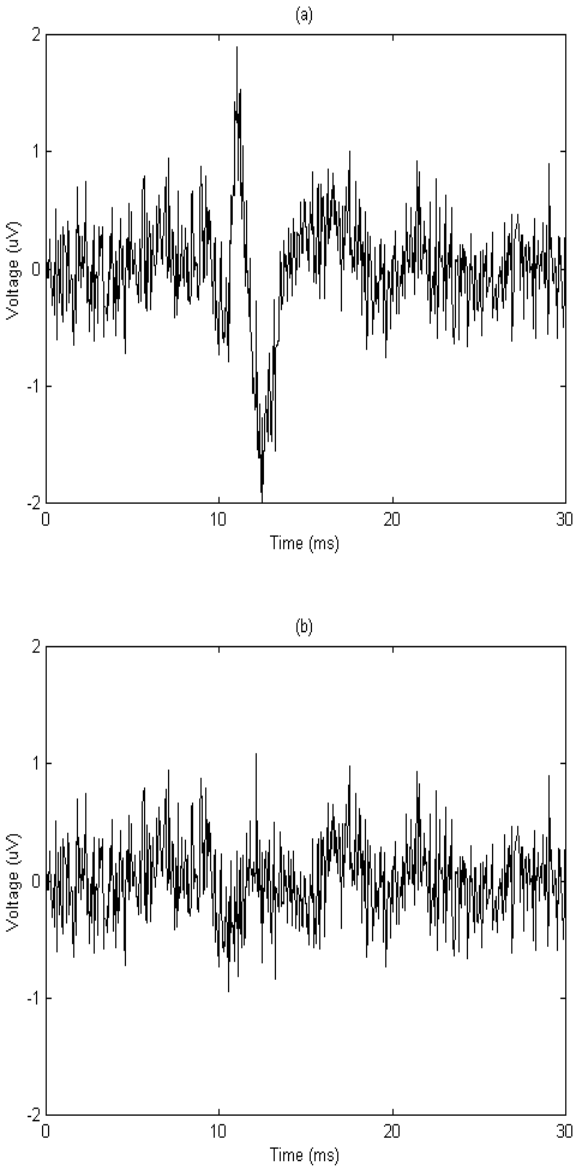

Fig. 2. Addition of white noise with a standard deviation of 0.3 μV to (a) the motor unit action potential of Fig. 1a and (b) the fibrillation potentials of Fig. 1c. The presence of ambient noise makes it impossible to determine the presence of the fibrillation potentials but the increased temporal coherence of the motor unit action potential allows it to be identified in the time domain even in the presence of noise.

potentials were uniformly distributed throughout the sequence. The spectral peak for each sequence overlaps in frequency. Despite the differing temporal distributions of the underlying basis vectors, each sequence results in nearly identical power spectrums.

V. Discussion

As shown in Fig. 1, increasing temporal dispersion among the muscle fibers of a motor unit results in an increasingly disorganized summed potential. The phase difference between the individual basis vectors results in cancellation of individual potential vectors and a smaller peak amplitude of the summed potential. Even though the sig-

nals of Fig. 1a and Fig. 1c lack similarity in the time domain, both signals are formed from the same set of basis vectors differing only in their temporal dispersion. The resultant signals have approximately the same spectral envelope and same frequency band of peak spectral energy.

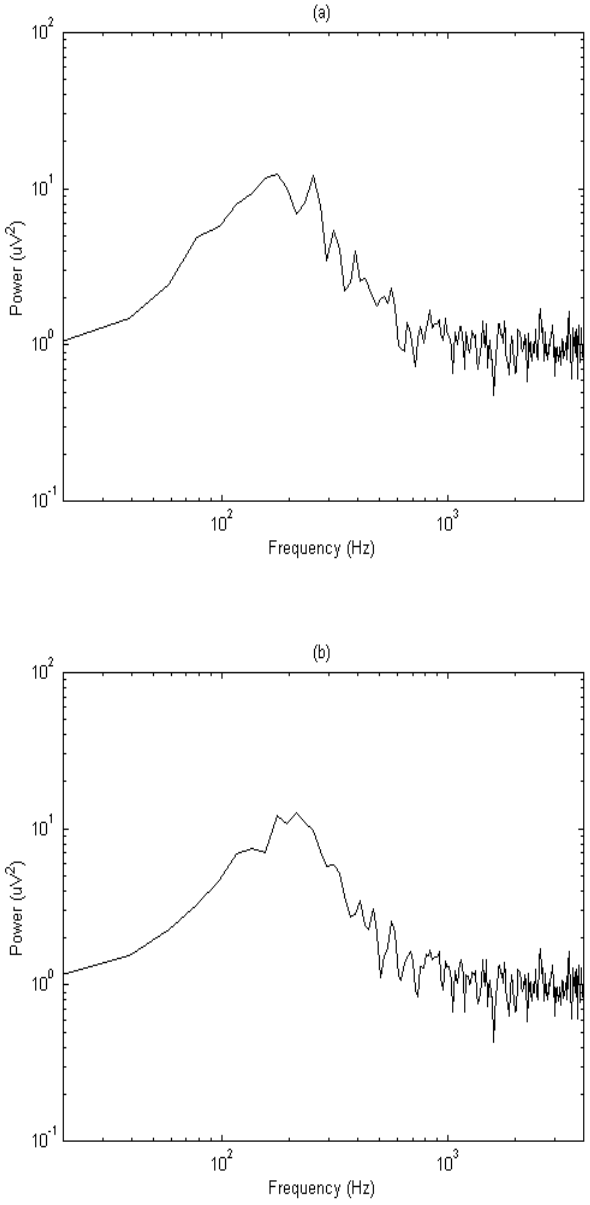

Fig. 3. Power spectral densities for (a) series of motor unit action potentials from 5000 fibers organized into motor units of approximately 200 fibers each firing over a one second period and (b) series of 5000 fibers firing individually during a one second period to model fibrillation potentials. The power spectral densities for each series share the same frequency band for peak spectral energy.

The small amplitude of fibrillations and their shared frequency band with MUAPs makes them impossible to detect in the presence of normal motor activity. Standard linear filtering techniques are unable to remove motor activity due to the spectral overlap with the signal of interest. However, filtering strategies taking advantage of the different temporal dispersion distributions of the MUAP and fibrillation potential basis vectors could possibly be implemented to remove MUAPs from recorded data sequences.

The determination of the presence of fibrillation potentials in a data sequence containing varying levels of normal motor activity represents a class of system identification problems where the signal of interest overlaps in frequency with a noise signal. A secondary characteristic of the signals, in this case the difference in temporal coherence of the signal basis vectors, is required to differentiate the noise signal from the signal of interest. Matched filters, taking advantage of the specific MUAP morphology, have the potential to remove motor activity while leaving intact the underlying spontaneous muscle activity signal.

VI. Conclusions and Future Work

The ability to non-invasively detect fibrillation potentials would allow for the development of improved neuromuscular diagnostics. Such diagnostics may result in lowered patienty anxiety, be useful in tracking patient response to treatment, and increase patient accessibility to diagnosis. Before such systems can be developed, however, filtering methods to remove normal motor activity must be implemented. Fibrillation potentials and normal motor activity share the same basis vectors resulting in spectral energy in overlapping frequencies bands making standard linear filtering ineffective. Future work will consist of utilizing the difference in temporal dispersion of the basis vectors as a means of developing filters to remove normal motor activity from surface recordings.

References

[1] S. Keller, A. Sandrock, and S. Gozani, "The non-invasive detection of spontaneous muscle activity," Submitted for Publication.

[2] J. Kimura, *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*, F.A. Davis Company, Philadelphia, second edition, 1989.

[3] M.J. Aminoff, *Electromyography in Clinical Practice*, Churchill Livingstone, Inc., New York, third edition, 1998.

[4] N. Dimitrova, "Model of the extracellular potential field of a single striated muscle fibre," *Electromyogr. Clin. Neurophysiol.*, vol. 14, pp. 53–66, 1974.

[5] I. Gath and E. Stålberg, "On the measurement of fibre density in human muscles," *Electroencephalography and Clinical Neurology*, vol. 54, pp. 699–706, 1982.

[6] P. Horowitz and W. Hill, *The Art of Electronics*, Cambridge University Press, New York, second edition, 1989.

[7] M. R. Neuman, *The Biomedical Engineering Handbook*, chapter Biopotential Electrodes, CRC Press, second edition, 2000.

[8] B. J. Roth, *The Biomedical Engineering Handbook*, chapter The Electrical Conductivity of Tissues, CRC Press, second edition, 2000.

[9] J. Belmar and C. Eyzaguirre, "Pacemaker site of fibrillation potentials in denervated mammalian muscle," *Journal of Neurophysiology*, vol. 29, pp. 425–441, 1966.

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